REMARKS

Claims 1-3, 5-33 and newly added Claim 34 are pending herein. Claim 4 has been cancelled without prejudice or disclaimer. Claims 3, 5-6, 8 and 14-33 stand withdrawn from consideration.

1. In the Office Action, the drawings filed on February 8, 2005, were objected to by the Examiner. However, as noted in a PTO communication dated December 1, 2005, this was an error. Therefore, no objection to the drawings is currently pending in the present application.

2. Claim 4 was objected to as encompassing non-elected subject matter. Claim 4 has been cancelled. This rejection is therefore moot and is respectfully requested to be withdrawn.

3. Claims 1-2, 4, 7-9 and 13 were rejected under 35 U.S.C. §112, first paragraph. The Examiner essentially stated that while the specification is enabling for detecting a disruption in normal cellular distribution of a G-protein receptor kinase 2/5 (GRK 2/5) in association with a transgenic model with early onset of Alzheimer's disease (AD), CRND8 mice, it does not reasonably provide enablement for disruption of all forms of GRK distribution and also for detecting all the Alzheimer's pathogenesis as broadly claimed. Further, the Examiner stated that while being enabling for detecting the disruption in normal distribution of GRK 2/5, the specification does not reasonably provide enablement for detecting the pathogenesis for AD by using soluble ß-amyloid

peptides in a diagnostic method.

Claim 1 has now been amended to recite a method of detecting early stages of Alzheimer's pathogenesis *in vitro* or in a transgenic model, which includes detecting a disruption in normal cellular distribution of a G-protein receptor kinase 5 (GRK5). Likewise, newly added Claim 34 recites a method of detecting a disruption in normal cellular distribution of a G-protein receptor kinase 5 (GRK-5) by using a peptide. It is respectfully submitted that the amended claims are fully supported by and are enabling by the present specification. Therefore, it is respectfully submitted that Claims 1-2, 7, 9-13 and 34 are in full compliance with §112.

The Examiner further stated that the specification fails to teach how GRK is associated with GPCR in contribution to the pathogenesis of AD or how GRK is a contributory factor for AD pathogenesis, and that the specification fails to disclose how the change of distribution (of the GRK) can be a determining factor for detecting the pathogenesis of AD. The Applicants respectfully disagree.

It is respectfully submitted that the specification discloses that the disrupted GRK 2/5 resulted in prolonged GPCR (i.e., thrombin receptors) signaling and potentiated inflammatory responses via the GPCRs. See Figures 3A-3G, 4A-4B, 5A-5C and 6A-6B, paragraphs [0027] - [0030], [0045] - [0048], and Examples 3-6 discussed in paragraphs [0051] - [0055].

CONCLUSION

For the foregoing reasons, it is respectfully submitted that Claims 1-2, 7, 9-13, and 34 are in condition for allowance. Withdrawal of all the rejections and objections and allowance of these claims are respectfully solicited.

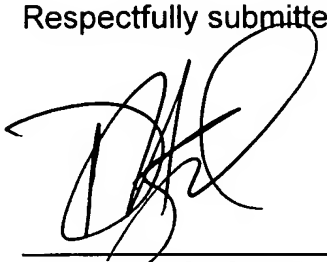
It is further respectfully submitted that withdrawn Claims 3, 5-6 and 8 are also allowable since generic Claims 1 and 7 are allowable.

Attached hereto is a check for $200.00 to cover the fee for one additional independent claim.

It is believed that no additional fee is due for this submission. Should that determination be incorrect, however, the Commissioner is hereby authorized to charge any deficiencies, or credit any overpayment, to our Deposit Account No. 01-0433, and notify the undersigned in due course.

Should the Examiner have any questions or wish to discuss further this matter, please contact the undersigned at the telephone number provided below.

Respectfully submitted,



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